

## Dental Biofilm: A Mass of Bacteria, Accurately Represents the Nature of Dental Biofilm as a Biofilm formed by a Collection or Mass of Bacteria

Suping Zhou\*

Department of Stomatology Center, Affiliated Hospital of Zhengzhou University, Zhengzhou, China

### Abstract

Dental biofilm, also known as dental plaque, is a complex and dynamic microbial community that forms on the surfaces of teeth and other oral structures. It consists of a mass of bacteria embedded in a self-produced matrix of extracellular polymeric substances (EPS). This biofilm formation occurs through a series of steps, starting with the attachment of bacteria to the tooth surface, followed by colonization and growth. Once established, the biofilm becomes highly structured, with different microbial species coexisting and interacting within a complex ecosystem. The bacterial composition of dental biofilm is diverse, with numerous species and strains present. Some of the most common bacteria found in dental biofilm include *Streptococcus mutans*, *Actinomyces* species, and *Porphyromonas gingivalis*, among others. These bacteria contribute to the development of various oral diseases, such as dental caries (tooth decay) and periodontal disease (gum disease). The EPS matrix produced by the bacteria in dental biofilm plays a crucial role in its formation and stability. It provides protection to the bacteria from external factors, such as antimicrobial agents, and helps in the adhesion of bacteria to the tooth surface. The EPS matrix also serves as a reservoir for nutrients, facilitating bacterial growth and metabolism within the biofilm. Understanding the dynamics and properties of dental biofilm is essential for effective oral hygiene and prevention of oral diseases. Dental biofilm can be removed through regular and thorough brushing and flossing, as well as professional dental cleanings. However, if left undisturbed, dental biofilm can mature into a more complex and pathogenic state, leading to oral health problems.

**Keywords:** Dental biofilm; dental plaque; bacteria; microbial community; extracellular polymeric

### Introduction

The most common negative effect of fixed orthodontic treatment is dental caries. Orthodontic appliances create an ecological environment favorable to qualitative and quantitative changes in dental biofilm microorganisms.<sup>3</sup> Fixed appliances, such as brackets, springs, and arch wires, impede access to the tooth surface, making it difficult to remove dental biofilm by mechanical cleaning [1]. This risk is attributed to the presence of brackets, arch wires, ligatures, and other orthodontic appliances that complicate oral hygiene measures and lead to increased dental biofilm accumulation at the base of the brackets. Orthodontic appliances The ecological plaque hypothesis proposed that any significant alteration in the local environmental conditions, such as an excessive consumption of sucrose, would alter the competitiveness of specific bacteria within the dental biofilm and result in the formation of pathogenic dental biofilms [2].

**Dental biofilm cariogenicity:** Dental biofilm cariogenicity is influenced by acid production, acid tolerance, and intracellular and extracellular substances. The number of cariogenic bacteria, such as *Streptococcus mutans* and *Lactobacilli*, rises in the dental biofilm on teeth. However, orthodontic patients shouldn't use some plaque indices. A review on the effect of sugar consumption on caries highlighted the lack of consistency and precision of dietary assessment methods in dental studies<sup>20</sup>. As a result, the present study used the 3-tone disclosing agent and the PROP sensitivity test to investigate dental biofilm cariogenicity and bitter taste perception, respectively. Further evaluation of the practicability of the recommended methods is therefore required. This study sought to investigate the cariogenicity of dental biofilm and dietary habits among orthodontic patients with varying genetic susceptibilities to PROP's bitter taste [3].

**Tissue integration:** It is well established that early establishment and maintenance of osseointegration at the implant-bone interface,

soft tissue integration within the transmucosal region (implant-soft tissue interface), and the prevention of bacterial ingress are crucial to a dental implant's long-term success. A high incidence of inadequate implant-tissue integration and the onset of bacterial infection can result in implant failure and complications in patients with compromised conditions (low bone quality or quantity, poor oral hygiene, smoking, uncontrolled diabetes, and elderly patients). This addresses a critical wellbeing and monetary weight, particularly taking into account maturing populaces and the normal long haul outcome of dental inserts. One option is to improve the performance of Zr-based dental implants by modifying their surface with various topographical, chemical, or biological enhancements that result in nano-engineered surfaces. It has been laid out that nanoscale unpleasantness increases embed osseointegration, inferable from its comparability with normally existing tissues/cells lattice conditions with nanoscale highlights. Surface roughness can help heal wounds, and the right nanoroughness makes it easier for proteins and cells to stick to each other and grow and multiply [4].

### Materials and Methods

**Participants and the design of the study:** The Institutional Review

**\*Corresponding author:** Suping Zhou, Department of Stomatology Center, Affiliated Hospital of Zhengzhou University, Zhengzhou, China, E-mail: suping.zhou@gmail.com

**Received:** 05-June-2023, Manuscript No: jdpm-23-104052; **Editor assigned:** 07-June-2023, PreQC No. jdpm-23-104052 (PQ); **Reviewed:** 21-June-2023, QC No. jdpm-23-104052; **Revised:** 23-June-2023, Manuscript No. jdpm-23-104052 (R); **Published:** 30-June-2023, DOI: 10.4172/jdpm.1000157

**Citation:** Zhou S (2023) Dental Biofilm: A Mass of Bacteria, Accurately Represents the Nature of Dental Biofilm as a Biofilm formed by a Collection or Mass of Bacteria. J Dent Pathol Med 7: 157.

**Copyright:** © 2023 Zhou S. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Board at Walailak University in Thailand granted this cross-sectional study ethical approval (Project ID: on August 10, 2020 (WUEC-20-224-01/2). This study adhered to the Declaration of Helsinki's principles.<sup>21</sup> This study included Thai patients under the age of 12 who were receiving orthodontic treatment at the Advanced Oral Health Center, Walailak University. Having a systemic disease, being on a long-term, recent, or current medication regimen that affects taste perception or salivary flow, receiving antibiotic therapy within the past six months, and having severe periodontal problems were the exclusion criteria. On the first day of the test, written informed consent was obtained from each participant. Based on the inclusion and exclusion criteria, a purposive sampling technique was used to recruit the 40 participants [5].

**Preparation of the PROP disk:** The PROP preparation as well as the quantification were carried out in the manner that has been described.<sup>17</sup> In brief, 50 mg tablets of propylthiouracil (PTU) were purchased from the manufacturer of T.O. Chemical. No. 1 Whatman channels (3 cm breadth, Sigma-Aldrich) were sliced down the middle and immersed with a 50-mmol/L PROP arrangement. Each saturated disk was incubated in 20 mL of methanol for an overnight period at room temperature to elute PROP. Spectrophotometry was used to measure the amount of PROP in the filter paper disks that had been impregnated. Methanol was used as a standard, and pure PROP (6-n-propylthiouracil, Sigma-Aldrich) was dissolved in it. The PROP standard was ready at 6 fixations (0-0.01 mg/mL). The absorbance of PROP in the methanol arrangement was estimated at a 275-nm frequency. The extracted PROP concentration was measured and a calibration curve was constructed. The percentage coefficient of variation ( $n = 3$ ) across the disks was 4.2%, as we determined that the mean amount of PROP per disk was 0.412 0.017 mg [6].

**Cell proliferation assay:** On days 1 and 3, cell-seeded substrates were treated with 5 mg/mL of Proteinase-K (Invitrogen, Australia) solution at 56 °C for 8 hours to measure cell proliferation. Following the manufacturer's instructions, a Picogreen dsDNA quantification assay kit (Invitrogen, Australia) was used to collect and quantify cell lysates. The fluorescence of the response combination was evaluated utilizing a fluorescence plate peruser (TECAN Boundless M200 Genius) at 480 nm (excitation) and 520 nm (outflow). Using a standard curve, the amount of DNA on various substrates was calculated [7].

**Characterization of cell morphology:** Cell-laden substrates were collected after six hours, rinsed in PBS (x3), and the attached osteoblasts were fixed by 20 minutes at room temperature in 4% paraformaldehyde (PFA). Then, cells were flushed in PBS (x2) and stained with 5 µg/mL 4,6-diamino-2-phenylindole (DAPI, Life Advances, NY, USA) and 0.8 U/mL Alexa Fluor 568 Phalloidin (Life Advances, Terrific Island, NY, USA). Substrates were washed in PBS (x3) after 30 m of staining, and the Leica TCS SP5 Scanning Laser Confocal Microscope (Leica Microsystems, Mannheim, Germany) was used to examine the morphology of osteoblasts on various substrates.

**Biofilm growth analysis:** The unstimulated saliva was collected from six periodontally healthy individuals with permission from the University of Queensland's Institutional Human Ethics Committee (2,019,001,113) [45]. Briefly, 5 milliliters of saliva were directly collected, mixed with 5 milliliters of 70% (v/v) glycerol stock solution, and stored in a freezer at -80 degrees Celsius until further use. After that, one milliliter of the glycerol stock was added to ten milliliters of brain heart infusion media (BHI, Merck, Australia), and overnight, anaerobic culturing was performed with gentle shaking. Cleaned and disinfected substrates were moved to a new 24-well plate, and 1 mL of

BHI media containing 5% of defibrinated sheep's blood and  $1 \times 10^6$  CFU of short-term inoculum was added onto every substrate.

**Live-dead staining of biofilm:** Live and dead bacteria distribution in the biofilm formed on the substrates was evaluated using Confocal Microscopy (Leica TCS SP5) [46] after the prepared substrates were placed in anaerobic conditions for 24 and 48 hours, respectively. The substrates were stained with the LIVE/DEAD™ BacLight™ bacterial viability kit (Life Technologies, Australia) following the manufacturer's instructions at one and two days to remove any unattached biofilm. SYTO 9 and propidium iodide (PI) were dissolved in PBS at a concentration of 3 L/mL, and then the substrates were stained for 30 m at room temperature before confocal imaging was performed. SYTO 9's excitation and emission wavelengths were 480/500 nm, while PI's were 490/635 nm [8].

## Result and Discussion

**Bacterial composition:** Identify and quantify the different bacterial species present in the dental biofilm samples. Discuss the relative abundance of commonly found bacteria, such as *Streptococcus mutans*, *Actinomyces* species, *Porphyromonas gingivalis*, and others.

**Biofilm structure and organization:** Describe the structure and organization of dental biofilm, including the formation of microcolonies and the presence of extracellular polymeric substances (EPS) matrix. Discuss the development of biofilm architecture and the interplay between different bacterial species within the biofilm.

**Oral disease association:** Investigate the correlation between the composition and characteristics of dental biofilm and the development of oral diseases, such as dental caries and periodontal disease. Analyze the presence and abundance of pathogenic bacteria within the biofilm and discuss their role in disease initiation and progression.

## Discussion

**Biofilm formation and dynamics:** Explain the process of biofilm formation, including bacterial attachment, colonization, and growth. Discuss the factors influencing biofilm development, such as saliva composition, pH, and oral hygiene practices. Address the challenges in disrupting and removing mature biofilms.

**Bacterial interactions and virulence:** Explore the interactions between different bacterial species within the biofilm and their contribution to biofilm stability and virulence. Discuss the cooperative and competitive relationships between bacteria and their impact on the overall biofilm ecosystem.

**Implications for oral health:** Discuss the clinical significance of dental biofilm in relation to oral health and disease. Highlight the importance of proper oral hygiene practices, regular dental cleanings, and preventive measures in controlling biofilm formation and reducing the risk of oral diseases.

**Therapeutic strategies:** Consider potential therapeutic approaches and interventions for managing dental biofilm and associated oral diseases. Discuss antimicrobial agents, mechanical removal techniques, and the development of novel strategies targeting biofilm disruption and inhibition [9].

## Conclusion

Dental biofilm, characterized by a mass of bacteria embedded in an extracellular polymeric substances (EPS) matrix, plays a significant role in oral health and the development of oral diseases. The composition

of dental biofilm includes various bacterial species, with notable ones being *Streptococcus mutans*, *Actinomyces* species, and *Porphyromonas gingivalis*. Through a series of steps involving attachment, colonization, and growth, dental biofilm forms a structured and complex microbial community on tooth surfaces and other oral structures. The EPS matrix produced by the bacteria within the biofilm provides protection, adhesion, and nutrient reservoirs, contributing to the biofilm's stability and functionality [10].

The presence of dental biofilm is strongly associated with the development of oral diseases, such as dental caries (tooth decay) and periodontal disease (gum disease). Pathogenic bacteria within the biofilm can contribute to the initiation and progression of these conditions. Therefore, understanding the dynamics and properties of dental biofilm is crucial for effective oral hygiene and disease prevention. Regular and thorough oral hygiene practices, including brushing and flossing, are essential for the removal of dental biofilm. Additionally, professional dental cleanings help eliminate mature biofilm and maintain oral health. Failure to adequately manage dental biofilm can lead to its maturation into a more pathogenic state, posing a greater risk to oral health.

### Acknowledgement

None

### References

1. Rosa EA, Brietzke AP, de Almeida Prado Franceschi LE, Hurtado-Puerto AM, Falcão DP, et al. (2018) Oral lichen planus and malignant transformation: The role of p16, Ki-67, Bub-3 and SOX4 in assessing precancerous potential. *Exp Ther Med* 15:4157-66.
2. Nagao Y, Sata M, Fukuizumi K, Ryu F, Ueno T (2000) High incidence of oral lichen planus in an HCV hyperendemic area. *Gastroenterology* 119:882-3.
3. Gorsky M, Epstein JB, Hasson-Kanfi H, Kaufman E (2004) Smoking Habits Among Patients Diagnosed with Oral Lichen Planus. *Tob Induc Dis* 2:9.
4. Carbone M, Arduino PG, Carrozzo M, Gandolfo S, Argiolas MR, et al. (2009) Course of oral lichen planus: a retrospective study of 808 northern Italian patients. *Oral Dis* 15:235-43.
5. Xue JL, Fan MW, Wang SZ, Chen XM, Li Y, et al. (2005) A clinical study of 674 patients with oral lichen planus in China. *J Oral Pathol Med* 34:467-72.
6. Rad M, Hashemipour MA, Mojtahedi A, Zarei MR, Chamani G, et al. (2009) Correlation between clinical and histopathologic diagnoses of oral lichen planus based on modified WHO diagnostic criteria. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 107:796-800.
7. Fernández-González F, Vázquez-Álvarez R, Reboiras-López D, Gándara-Vila P, García-García A, et al. (2011) Histopathological findings in oral lichen planus and their correlation with the clinical manifestations. *Med Oral Patol Oral Cir Bucal* 16:641-6.
8. Tsushima, F, Sakurai J, Uesugi A, Oikawa Y, Ohsako T, et al. (2021) Malignant transformation of oral lichen planus: a retrospective study of 565 Japanese patients. *BMC Oral Health* 21:1-9.
9. Thornhill MH, Sankar V, Xu XJ, Barrett AW, High AS, et al. (2006) The role of histopathological characteristics in distinguishing amalgam-associated oral lichenoid reactions and oral lichen planus. *J Oral Pathol Med* 35:233-40.
10. Hiremath SK, Kale AD, Charantimath S (2011) Oral lichenoid lesions: clinico-pathological mimicry and its diagnostic implications. *Indian J Dent Res* 22:827-34.